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Review article

## An update on adolescent sleep: New evidence informing the perfect storm model<sup>☆</sup>

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## ABSTRACT

The maturation of sleep regulatory systems during adolescence in combination with psychosocial and societal pressures culminate in a “Perfect Storm” of short and ill-timed sleep and the associated consequences for many youngsters. This model, first described by Carskadon in 2011, guides our current thinking of adolescent sleep behavior. Since the original description, the field has moved forward with remarkable pace, and this review aims to summarize recent progress and describe how this new work informs our understanding of sleep regulation and sleep behavior during this developmental time frame.

A 2011 review paper (Carskadon, 2011) proposed that maturation of bioregulatory mechanisms in concert with psychosocial factors resulted in a “perfect storm” of short, ill-timed, and inadequate sleep in many teens. Two biological systems undergo modifications during adolescence. First, is the sleep/wake homeostatic process, which not only marks sleep recovery and restoration, but also signifies the speed with which the “pressure” for sleep and resulting deficits build across the day. Findings indicated that for more mature adolescents the recovery process did not accelerate, whereas the sleep pressure accumulation process decelerated, from which Carskadon inferred an ease of staying awake longer. The second process in this biological regulatory equation is the circadian timing system, which dictates the timing of many physiological and behavioral rhythms, including alertness and sleep propensity. Findings reviewed by Carskadon indicated a delayed shift in the intrinsic rhythmic system as adolescents mature, hence pushing alertness and bedtimes later into the evening and night and rising later into the morning. Data from the behavioral and psychosocial literature were sparse, yet led to a conclusion that evening light from devices with screens may activate the phase-delaying component of the circadian timing mechanism. Opposing these factors that drive the temporal placement of sleep later were such early morning events as sports practices and an early starting time for school.

In brief, the field has moved forward with remarkable pace since the earlier review was published (Carskadon, 2011), and we aim to detail this progress following a brief overview.

With regard to the homeostatic sleep “drive” process, for example, longitudinal studies have confirmed that the dissipation of sleep pressure does not change across adolescent development (Campbell et al., 2011; Tarokh, Carskadon, & Achermann, 2012). Recommended sleep length guidelines have been proposed by the National Sleep Foundation and the American Academy of Sleep

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Medicine (Hirshkowitz et al., 2015; Paruthi et al., 2016), in both cases based upon expert judgment of the literature. We describe below more recent experimental findings that may provide evidence-based guidance on sleep duration for adolescents, concluding that about 9–9.25 h a night is required for cognitive function/attention (Short, Weber, Reynolds, Coussens, & Carskadon, 2018) and for emotional regulation (Fuligni, Bai, Krull, & Gonzales, 2017). Yet, several studies from around the globe continue to report average sleep durations less than these healthful amounts. For example, in a recent meta-analysis of studies in which sleep behavior was measured from actigraphy in children and adolescents ranging in age from 3 to 18 years, total sleep time decreased with age, with pooled mean estimates in 12–18 year olds of about 7 h on school nights (Galland et al., 2018). The disparity between sleep duration measured in the laboratory (9.25 h) and the home environment (7 h) reinforce an interplay between bioregulatory mechanisms and psychosocial factors.

We note with regard to the circadian timing system, a new study of a juvenile nonhuman mammal species, the marmoset, shows a delay of activity rhythms (Melo, Goncalves, Menezes, & Azevedo, 2016). This study seems to support suggestions of the earlier review that findings from nonhuman vertebrates help to substantiate the biological underpinnings of adolescent phase delay findings. Other new work detailed below has constructed a phase response curve (PRC) to light in late and post-pubertal adolescents and found a pattern that is contrary to predictions about how adolescents differ from adults (Crowley & Eastman, 2017; Crowley, unpublished). These data suggest that the circadian phase delay of late adolescence is not due to difference in circadian phase-shifting responses to light. Furthermore, an investigation of circadian period also showed no difference between older adolescents and adults (Crowley & Eastman, 2018). When combined with evidence for increased sensitivity to evening light (Crowley, Cain, Burns, Acebo, & Carskadon, 2015), as well as longer measured intrinsic period (Carskadon, Barker, Crowley, Rupp, & Van Reen, 2017) both in younger adolescents, a growing sense is emerging that biological changes affecting circadian timing arise earlier in development than previously predicted. One longitudinal study of circadian phase in adolescents (Crowley et al., 2014) proposed a model that links the changes to sleep/wake homeostasis to circadian timing in a manner that helps to explain delayed sleep patterns as adolescents develop.

Materials described below extend the previous review paper significantly by detailing recent findings regarding adolescent sleep behaviors and nonbiological factors that affect sleep, the most prominent of which is the starting time of schools. A major advance with potential to influence the psychosocial landscape of adolescent sleep has been a forceful public health outcry and associated recommendations for delaying the starting times of schools as a means to alleviate both short duration sleep as well as ill-timed and irregular sleeping patterns. The American Academy of Pediatrics (Adolescent Sleep Working Group, Committee on Adolescence, & Council on School Health, 2014), Centers for Disease Control (Wheaton, Chapman, & Croft, 2016), and the American Academy of Sleep Medicine (Watson et al., 2017) have made the case, based largely on small cross-sectional studies linking school start time to such issues as driving crashes and large epidemiological studies that place short sleep duration of adolescents as a risk factor for serious behavioral and emotional consequences. This message has been taken up by a number of advocacy groups, including Start School Later ([www.startschoollater.net](http://www.startschoollater.net)), who have undertaken lobbying efforts to implement change. Although these efforts seem to target high school age students, the issue is relevant to and targets middle school students as well (cf. Temkin, Princiotta, Ryberg, & Lewin, 2018). As shown in the systematic review by Wheaton and colleagues (Wheaton et al., 2016), much of the literature in support of delaying school start times has been gathered in the United States, though data from other regions including Hong Kong, China, Israel, Turkey, Switzerland, Spain, and New Zealand also show better sleep outcomes when schools start later. In a recent report from England (Kelley, Lockley, Kelley, & Evans, 2017), a school's start time was shifted from 8:50 am to 10:00 am for 2 years and then shifted back to 8:50 a.m. due to a change in local education administrators. This naturalistic experiment suggested that the recommended 8:30 a.m. school start time may be too early as the number of sick days decreased and performance on general examinations improved during the 2 years in which school started later (Kelley et al., 2017). Finally, Lo et al. (2018) showed increased total sleep and self-reported well-being 1 month following a 45-min delay in school start time in female students at a Singapore high school, with changes sustained at a 9-month follow-up. Authors discussed how in a culture where academic achievement is high priority, delaying school start time was feasible, sustainable, and positively accepted by parents, students, and teachers.

A pattern of restricted sleep on school nights comes from the factors highlighted above that impinge on both ends of the sleep duration equation: bedtimes and wake-up times. Bedtimes on school nights shift later as youngsters transition through adolescence, while rise times remain stable or become earlier. While these age-related sleep timing changes across adolescence have been reviewed previously (Crowley, Acebo, & Carskadon, 2007; Gradisar, Gardner, & Dohnt, 2011), we describe below a more recent longitudinal study in the United States of two age cohorts spanning the second decade (9–19 years) that confirms these findings: sleep onset times measured from actigraphy shifted later as an adolescent got older, while wake-up times shifted earlier as an adolescent transitioned from middle school to high school (Crowley et al., 2014). Interestingly, this paper showed that wake-up times shifted later as an adolescent transitioned out of high school, reflecting the societal pressure of school start time being lifted and wake-up times being less constrained.

Our introduction provides a quick overview and gist of the newer findings that now inform the *Perfect Storm* model. The details that follow allow for a more in-depth review of adolescent sleep biology and behavior.

## 1. Sleep regulation

We begin with a quick overview of the guiding principles that structure the approach to assessing sleep regulation across adolescence. The Two-Process Model, first proposed by Borbely (1982), with its later refinements (Achermann, Dijk, Brunner, & Borbely, 1993; Borbely & Achermann, 1999; Borbely, Achermann, Trachsel, & Tobler, 1989; Daan, Beersma, & Borbely, 1984) and variations (Dijk & Czeisler, 1995; Edgar, Dement, & Fuller, 1993; Phillips & Robinson, 2007; Phillips, Chen, & Robinson, 2010) continues to guide our hypotheses and inform our understanding of developmental sleep regulatory changes that may explain sleep/wake

behavior during development. The model's two components consist of an approximate 24-h (circadian) timing system, the brain center of which has been localized to the suprachiasmatic nucleus of the hypothalamus, and a sleep-wake pressure (homeostatic) system, for which a neuroanatomical locus still remains unknown. The genetically-regulated central circadian clock within the brain signals fluctuations of more or less sleep propensity across the 24-h day regardless of prior sleep/wake duration. By contrast, the homeostatic sleep system favors sleep as wake is extended and favors wakefulness as sleep is prolonged. The homeostatic system is dependent on prior sleep/wake conditions and not the time of day. These two systems interact to regulate sleep duration and timing. As described in Carskadon's original review paper (Carskadon, 2011), sleep homeostasis and circadian physiology are altered as youngsters progress through adolescence and partly explain developmental changes in sleep behavior.

### 1.1. Sleep homeostasis during adolescence

Our fundamental understanding of the developmental changes to the sleep homeostatic system during adolescence has not been altered by new studies; however, a handful of recent studies have reinforced and expanded upon previous findings since 2011. Inferences regarding the sleep homeostatic system are primarily derived from the incidence and amplitude of low-frequency high amplitude waves in the sleep EEG called slow waves. The mathematical quantification of these waves, called slow wave activity, shows homeostatic behavior, increasing during waking and dissipating during sleep (Achermann & Borbely, 2017). Slow wave activity is sensitive to prior sleep/wake history and prolonged bouts of waking show a dose-dependent increase in this metric; thus slow wave activity has long been used as a measure of sleep's homeostatic process.

Cross-sectional studies modeling the accumulation of sleep pressure have shown that sleep pressure builds more slowly in more mature adolescents (i.e., post pubertal) as compared to younger (pre-/early pubertal) adolescents (Jenni, Achermann, & Carskadon, 2005). The authors hypothesized that the slower accumulation of sleep pressure allowed older teens to better withstand increased sleep pressure and thus delay bedtimes (Jenni et al., 2005). Indeed, in a study examining the speed of falling asleep following prolonged waking, pre-/early pubertal adolescents (mean age = 11.1 years) fell asleep faster than post-pubertal adolescents (mean age = 13.9 years) 14.5 and 16.5 h after waking (Taylor, Jenni, Acebo, & Carskadon, 2005). Specifically, when both groups were woken at 8:00 a.m., post-pubertal adolescents tended to take longer to fall asleep from 10:30 p.m. to 2:30 a.m.

On the other hand, longitudinal and cross-sectional studies modeling the *dissipation* of sleep pressure based on sleep EEG slow wave activity have shown that the rate at which sleep pressure is dissipated does not change across adolescent development (Campbell et al., 2011; Gaudreau, Carrier, & Montplaisir, 2001; Jenni & Carskadon, 2004; Jenni et al., 2005; Tarokh et al., 2012). The stability in the dissipation rate of sleep pressure implies that sleep need does not change across this developmental period, a finding that is in line with experimental evidence on sleep need in adolescence. Early evidence that sleep need does not change at the transition to adolescence came from a seminal study in which adolescents aged 10–17 years given a 10-h sleep opportunity slept on average 9.25 h irrespective of age or maturational stage (Carskadon, 1982).

This conclusion that the need for sleep does not diminish across adolescence is bolstered by two recent studies that examine behavioral outcomes rather than the sleeping EEG. In one, Short et al. (2018) computed the “dose response” of adolescents (ages 15–17 years) across 5 nights of 5, 7.5, or 10 h of time in bed. Their computation estimated that the median sleep need of adolescents to sustain waking vigilance and alertness is 9.3 h, compared to 8.16 h estimated for adults by Van Dongen, Maislin, Mullington, and Dinges (2003). A field study of over 300 adolescents (ages 13–19 years) with daily self-reported measures of sleep, distress, and externalizing and internalizing symptoms concluded that the overall “optimum” sleep for daily mood was centered at about 9 h across the sample (Fuligni et al., 2017). This study showed a modest, statistically significant decline in optimum sleep need measured with these outcomes across this age range. Both studies echo the earlier work based on multiple sleep latency test (i.e., measuring the time taken to fall asleep on multiple occasions) findings, which concluded a stable sleep need of about 9 h a night across adolescence (Carskadon, Orav, & Dement, 1983).

In addition to studies modeling the regulation of the homeostatic system during adolescence, a number of recent studies have examined the impact of sleep restriction on slow waves. One such cross-sectional study by Campbell and colleagues (Campbell, Kraus, Burright, & Feinberg, 2016) found that compared to a 10-h condition, reducing time in bed to 8.5 or 7 h for four nights did not impact slow wave activity in adolescents between the ages of 9.9–14.0 years when the same amount of non-REM sleep was included in the analysis. Another study which employed markedly more sleep restriction of five nights of 5 h of time in bed in a sample of late adolescents (15–19 years) found substantial build-up of sleep pressure over the five days as indexed by slow wave energy (Ong, Lo, Gooley, & Chee, 2017). Slow wave energy takes into account the cumulative amount of slow wave activity across the entire sleep period, and therefore differs from measures of slow wave activity averaged across a given time interval. Interestingly, two nights of recovery sleep consisting of 9 h of time in bed (akin to a weekend sleep-in) were insufficient to compensate for the lost slow wave energy over the restriction protocol (Ong et al., 2017).

The above-mentioned studies (Campbell et al., 2016; Ong et al., 2017) are difficult to compare directly given the differences in the sleep restriction protocols and the metrics used to measure sleep homeostasis. Another important difference between the studies is the different ages studied (i.e., early versus late adolescence) as current evidence suggests that the rebound in slow wave activity following sleep deprivation/restriction undergoes maturation across the adolescent period. For example, a study in humans found that pre-/early pubertal adolescents showed a proportionally smaller increase in slow wave activity following one night of sleep deprivation as compared to post-pubertal teens (Jenni et al., 2005). Similar findings were reported in a study of homeostasis in adolescent mice, which found an increase in slow wave activity after an acute sleep restriction (four hours) only in mice near the midpoint of adolescence (Nelson, Faraguna, Zoltan, Tononi, & Cirelli, 2013). This has led to the hypothesis that a ceiling effect may prevent slow wave activity from building in the younger brain (Jenni et al., 2005; Nelson et al., 2013). This diminished ability to

recover from sleep restriction/deprivation with a rebound in slow wave activity, in combination with the faster build-up of sleep pressure may be the reason why young adolescents and mice have difficulty staying awake during periods of prolonged wakefulness (Jenni et al., 2005). Indeed, in the study of adolescent mice, those in the early phase of adolescence made 21 sleep attempts during the four-hour deprivation phase as compared to seven attempts for the more mature adolescents (Nelson et al., 2013). Therefore, the physiological response to sleep restriction may change progressively across adolescent development and has implications for the ability of the brain to recover from sleep loss.

To summarize, there are two arms to the sleep homeostatic system: one which is associated with the rate at which sleep pressure builds during waking periods and the other is a reflection of the rate of dissipation of sleep pressure. During adolescent development, sleep pressure builds more slowly allowing older teens to delay their bedtimes. On the other hand, the rate at which sleep pressure is dissipated does not change, thus sleep need does not change across the adolescent years (i.e., remains stable around 9.25 h).

### 1.2. Circadian timing during adolescence

Early studies suggested that the central circadian clock shifts later (phase delay) with the progression of puberty, findings that were inferred from subjective measures initially (Andrade, Benedito-Silva, Domenice, Arnhold, & Menna-Barreto, 1993; Carskadon, Vieira, & Acebo, 1993) and then later confirmed with objective measures of circadian phase using salivary melatonin (Carskadon, Acebo, & Jenni, 2004; Carskadon, Acebo, Richardson, Tate, & Seifer, 1997). The circadian phase delay seen in humans is also observed in nonhuman mammals, including rhesus monkey, degu, laboratory rat, laboratory mouse, and the fat sand rat (Hagenauer, Perryman, Lee, & Carskadon, 2009). A more recent study adds to this list as it shows delayed activity rhythms in juvenile marmosets relative to their adult parents (Melo et al., 2016), and further substantiates the biological underpinnings of adolescent phase delay findings in humans.

Two primary hypotheses that could explain the puberty-related circadian delay of adolescents have guided recent work in this area. The first hypothesis posited that the endogenous circadian period – the internal day length of the circadian system – may lengthen over the course of puberty. The majority of adult humans show an intrinsic circadian period a little longer than 24 h (Duffy & Wright, 2005); for example, the average ranges from about 24.2 to 24.3 h (Burgess & Eastman, 2008; Czeisler et al., 1999; Duffy, Rimmer, & Czeisler, 2001; Duffy et al., 2011; Eastman, Molina, Dziepak, & Smith, 2012; Eastman, Suh, Tomaka, & Crowley, 2015; Eastman, Tomaka, & Crowley, 2017; Kitamura et al., 2012; Lazar et al., 2012; Smith, Burgess, Fogg, & Eastman, 2009; Wright, Hughes, Kronauer, Dijk, & Czeisler, 2001). If the system was left to run at its own pace (“free run”), sleep/wake patterns of those with circadian periods longer than 24 h would progressively get later and later each day. The process of entrainment (synchronizing internal rhythms with the external 24-h day) occurs primarily through light/dark exposure. The length of the free-running circadian period predicts how the circadian system entrains to the 24-h day (Burgess & Eastman, 2008; Duffy, Dijk, Hall, & Czeisler, 1999; Wright, Gronfier, Duffy, & Czeisler, 2005; Wright et al., 2001); a longer circadian period is associated with more owl-like evening tendencies, whereas a short circadian period is associated with larkish morning-type tendencies (Duffy et al., 2001). A lengthening of the endogenous circadian period would be consistent with the evening-type sleep/wake behavior of older more mature adolescents than say their pre-pubertal siblings or their parents. Preliminary evidence from animal (McGinnis, Lumia, Tetel, Molenda-Figueira, & Possidente, 2007) and human (Carskadon & Acebo, 2005) studies supported this hypothesis as these data suggested that adolescents have a longer circadian period than adults. A recent study by Crowley and Eastman do not support this hypothesis, however, as they show similar free-running circadian periods in late- and post-pubertal adolescents (Tanner stage 4 or 5; 14.3–17.8 years) compared to adults (30.8–45.8 years) when run in the same laboratory protocol (Crowley & Eastman, 2018). In this study, both age groups showed an average circadian period of about 24.2 h. Similar to previous studies in adults (Eastman et al., 2012; Eastman et al., 2017; Smith et al., 2009), ancestry differences were noted; free-running circadian period was shorter in African-American participants compared to those of other ancestries (mostly White), but this was primarily driven by the adult group as the mean ancestry differences in the adolescents was smaller by comparison. Sex differences in both age groups were also noted, though not at a statistically reliable level. These recent results would indicate that late and post-pubertal adolescents have adult-like free-running circadian periods, and therefore, do not provide support for a coincident change in free-running circadian period and sleep behavior during late adolescence. New data from the Carskadon laboratory (Carskadon et al., 2017) seems to indicate that changes to free-running circadian period may be occurring earlier in development, though analyses that consider ancestry and sex differences in these developmental patterns are needed.

The second mechanism proposed to explain a delayed circadian phase with pubertal development was an altered circadian response to light. The system's response to light is phase-dependent and predictable; in general, light exposure to the eyes in the evening or first part of habitual sleep shifts the circadian system later (phase delay) and light exposure in the second half of habitual sleep or shortly after waking shifts circadian rhythms earlier (phase advance). These responses are experimentally derived and illustrated as a phase response curve (PRC) to light. A delayed circadian system as seen in older adolescents could be explained by a blunted response to morning phase advancing light, an exaggerated response to evening phase delaying light, or a combination of both. Crowley et al. (2015) tested these hypotheses in a group of pre- to post-pubertal adolescents aged 9.1–15.9 years. Circadian sensitivity to light was assessed using melatonin suppression in response to 0, 15, 150, or 500 lux of light shown in the evening (between 23:00 – midnight) or the morning (03:00–04:00). In the evening, early to mid-pubertal adolescents (Tanner Stage 1–3) showed greater sensitivity to light compared to the late and post-pubertal adolescents (Tanner Stage 4 and 5), which contradicted the original evening light sensitivity hypothesis. Indeed, the younger group was incredibly sensitive to evening light such that responses were observed in light levels (~15 lux) far less than normal room lighting in most homes (~100 lux). The morning light sensitivity hypothesis showed some support; 500 lux of light initially suppressed melatonin more in the early- to mid-pubertal group compared to

the late and post-pubertal group, but this difference was not statistically reliable ( $p = .06$ ) and percent suppression was similar between groups at the end of the 1-h light exposure. These data, which showed no sex differences, suggested an overall reduction of the circadian clock's sensitivity to light, particularly in the evening as adolescents mature. Taken together with findings showing robust evening melatonin suppression in young children ( $4.3 \pm 1.1$  years) (Akacem, Wright, & LeBourgeois, 2018) and greater melatonin suppression in the evening in school-aged children ( $7.4 \pm 1.8$  years) compared to their parents ( $41.2 \pm 4.8$  years) (Higuchi, Nagafuchi, Lee, & Harada, 2014), this decline of evening light sensitivity is likely an age-related phenomenon and not related to puberty.

Melatonin suppression is one method to assess circadian light sensitivity; however, phase shifts (changes in the timing of the circadian rhythm) in response to light may be a more relevant outcome to test the hypothesis that adolescent sleep behavior is associated with altered responses to light. In particular, evening light may produce larger phase delay shifts as adolescents mature or phase advance shifts are attenuated in response to morning light. As reviewed previously (Carskadon, 2011), one animal study of female mice provides some support for this hypothesis as pre-pubertal (49 days) animals delayed more in response to a 15-min 150-lux light pulse compared to adults (140 days) (Weinert & Kompauerova, 1998). In an attempt to test this hypothesis in humans, Crowley and Eastman (2017) constructed a phase response curve to light in late and post-pubertal (Tanner Stage 4 and 5) adolescents aged 14.3–17.8 years. The adolescent phase response curve to light showed a predictable pattern with the largest delay shifts occurring in the hours straddling habitual bedtime, and the largest advance shifts occurring in the hours straddling habitual wake-up time. Moreover, the phase delay and phase advance regions of the adolescent phase response curve were symmetrical, suggesting that the phase delay shift in response to bright light is not exaggerated, and the phase advance response to bright light is not attenuated as predicted, in late- and post-pubertal adolescents. Also, inconsistent with previous animal data, the older adolescent phase response curve does not differ from a phase response curve constructed in adults (30–45 years) using the same protocol (Crowley, unpublished). Whether phase shift responses of older adolescents differ from their younger peers remains unknown.

These recent studies (Crowley & Eastman, 2017; Crowley et al., 2015) do not support the hypothesis that the circadian system of older adolescents are intrinsically more sensitive to evening light. What is more likely is the opportunity for light exposure in the evening increases as adolescents get older. Crowley et al. (2014) found that young adolescents (9–13 years) fell asleep about 1 h after the onset of their biological night, defined by the secretion of melatonin. Older adolescents (15–18 years), however, fell asleep 2 h into their biological night. The authors proposed that this difference in when they fell asleep with respect to the onset of melatonin secretion is due to a slowed accumulation of waking homeostatic sleep pressure allowing older adolescents to stay awake later into their biological night. Recent modeling work provides support for this hypothesis (Skeldon, Derks, & Dijk, 2015).

Changes to sleep physiology propping up evening alertness also increases the likelihood of light exposure later into the evening, a time when the circadian system is particularly sensitive to phase delaying light (Crowley & Eastman, 2017). Not to mention, an increasing number of adolescents access screens at night before bedtime (Gradisar et al., 2013; Hale et al., 2018), and growing evidence suggests that prolonged screen light exposure (i.e.,  $\geq 1.5$  h) can feed back into the circadian and homeostatic sleep systems (Cajochen et al., 2011; Wood, Rea, Plitnick, & Figueiro, 2013; van der Lely et al., 2015) to reinforce this evening alertness. It should be noted, however, that the assumption to avoid bright screens in the hour before bed has not yet shown to significantly attenuate the natural rise in melatonin (Wood et al., 2013; van der Lely et al., 2015) nor meaningfully influence the subsequent sleep of adolescents (Heath et al., 2014; van der Lely et al., 2015). Two meta-analyses of the effect of screen time on young people's sleep have been conducted since the 2011 Carskadon review (Bartel, Gradisar, & Williamson, 2015; Carter, Rees, Hale, Bhattacharjee, & Paradkar, 2016). When considering only portable interactive devices (i.e. smartphones, tablets) there is a two-fold increased effect on inadequate sleep (Carter et al., 2016). Yet when considering all devices, the associations with sleep duration are small (Bartel et al., 2015). Nevertheless, extrinsic alerting factors can interact with bioregulatory systems to perpetuate a cycle of delayed sleep timing and restricted sleep duration.

In sum, new data suggest that changes in central circadian physiology may be occurring earlier in development, while older adolescents are showing adult-like circadian physiology. It is still unclear whether the described changes in circadian physiology early in development provide the impetus for a delayed system in the later years of adolescence. Persistence of delayed and shortened sleep as adolescents mature may be ascribed to bioregulatory sleep changes and environmental factors that both reinforce evening alertness and delayed sleep onset.

## 2. Schools start times, cognitive functioning, and academic performance

Bioregulatory and psychosocial factors together create a deficient environment for adolescents' sleep health and the consequences that follow (Carskadon, 2011). Negative health outcomes include sleepiness mood disturbances, mental health difficulties, behavioral problems, substance use and abuse, weight gain, accidents including motor vehicles, and immune system challenges. School-related negative consequences include inattention, school absenteeism and tardiness, learning and memory difficulties, and poor academic grades. Likewise, a number of psychosocial factors affect sleep patterns in adolescents and contribute to the delay in sleep timing and insufficient sleep including school start times, socioeconomic status, digital media use, social engagements, and caffeine intake (Calamaro, Mason, & Ratcliffe, 2009; LeBourgeois et al., 2017; Ludden & Wolfson, 2010; Marco, Wolfson, Sparling, & Azuaje, 2011; Minges & Redeker, 2016). With one of the hallmark environmental constraints for adolescent sleep being the starting time of school, and keeping in mind that, on average, children spend 1000 h of instructional time per year in school (13,000 h from kindergarten through 12th grade), in the section below we review the research on school start times (constraint) and academic/cognitive functioning (consequence) associated with insufficient, delayed, and erratic sleep.

Early school schedules significantly contribute to insufficient sleep for adolescents in middle and high school. In a now hallmark

study, Carskadon and colleagues examined sleep, sleepiness, and circadian rhythms in adolescents in a school district that advanced school start times from 8:25a.m. to 7:20a.m. from 9th (end of junior high school) to 10th grade or high school (Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998). Using actigraphically estimated sleep, findings demonstrated that although the adolescents didn't go to sleep earlier, they woke up significantly earlier on school days when school start times were advanced, 10th versus 9th grade, and therefore obtained less sleep. Furthermore, comparing a 9th grade overnight assessment vs. 10th grade, these adolescents demonstrated increased daytime sleepiness, shortened sleep onset latency, and nearly 50 percent of the 10th graders had REM sleep on the first morning (8:30am) assessment within only 10 or 15 min of sleep onset, (i.e., a finding seen in a narcolepsy diagnosis (Mignot et al., 2006)). Moreover, this study revealed that the melatonin onset phase (i.e., the circadian timing system) for these adolescents was 40 min later in 10th versus 9th grade (Carskadon et al., 1998). In other words, these adolescents biologically needed to be sleeping at the time that they were required to be in school ready to tackle any number of high school subjects such as calculus, literature, and chemistry. Over the last 25 years, more attention has been directed toward delaying middle and high school start times because many school districts in the U.S. and around the world start the school day earlier as children progress through school, thus requiring adolescents to get up at an early hour juxtaposed to their bedtimes, circadian phases, and need for sleep. Since Carskadon and colleagues' study, school start time outcome research from a range of disciplines and methodologies: circadian rhythms to economics, and across different countries, have investigated the impact of delaying school start times on adolescents' health and academic performance as well as on the ramifications for school districts (Hafner, Stepanek, & Troxel, 2017; Lee, Nolan, Lockley, & Pattison, 2017; Owens, Wang, Lewin, Skora, & Baylor, 2016; Wahlstrom, 2016).

Wahlstrom's study of over 18,000 high school students was one of the first to demonstrate that delaying school start times increased attendance, improved high school enrollment, and showed some improvement in grades (Wahlstrom, 1999, 2002). Moreover, these students had similar bedtimes to students from other earlier starting schools, despite the delay in school start time. Moreover, on average, students at the later starting high schools got almost one hour more sleep each school night. Since Wahlstrom's study, numerous studies have examined various aspects of the impact of delaying school start times on adolescents' sleep, health, daytime functioning, and academic performance along with the economic and policy implications (Adolescent Sleep Working Group et al., 2014; Hafner et al., 2017; Troxel & Wolfson, 2017). Three recent reviews document the body of work conducted over the last 25 years on school start times and adolescents' sleep. Using a meta-analysis approach, Minges and Redeker and Bowers and Moyer examined the effects of delayed school start time on adolescents' sleep, health, and academic outcomes (Bowers & Moyer, 2017; Minges & Redeker, 2016). Based on Minges and Redeker's meta-analysis, school start times were delayed 25–60 min, and correspondingly, total sleep time increased from 25 to 77 min per weeknight. In addition, studies reported reduced daytime sleepiness, depression, caffeine use, and tardiness (Minges & Redeker, 2016). Bowers and Moyer, similarly, concluded that later starting school times are associated with longer sleep durations, less daytime sleepiness, and tardiness to school (Bowers & Moyer, 2017). Additionally, Wheaton and colleagues (Wheaton et al., 2016) systematically reviewed 38 studies, captured through PubMed and Scopus that focused on the association between school start times, sleep, and other behavioral outcomes. They concluded that there is significant evidence that delaying school start time increases school-night sleep duration by at least 30 min, primarily by delaying rise times; and that later start times generally correspond to improved attendance, less tardiness, decreased incidence of falling asleep in class, fewer motor vehicle crashes, and improved grades (Wheaton et al., 2016).

Understanding the impact of delayed school start times on academic performance specifically, as well as related factors such as tardiness and absenteeism is timely. Yet, as Wheaton and colleagues discuss, it is challenging to assess for several reasons: 1) grading is not standardized and varies by subject, teacher, and school; 2) achievement tests vary across school districts and standardized tests (e.g., Secondary School Admission Test (SSAT), Preliminary Scholastic Aptitude Test (PSAT), or the SAT) are not taken by all students; 3) variables associated with struggling in school are complex and multifaceted; and 4) it is challenging to assess the impact of delaying school start times for high achieving students as they have less room for improvement in this realm even though there are overall health benefits (Wheaton et al., 2016). Nonetheless, studies conclude that later school start times have a positive impact on academic performance and behavior. For example, in Wahlstrom's more recent multisite study of eight school districts, researchers concluded that the majority of schools reported an increase in GPA with a delay in school start times (Wahlstrom et al., 2014). Another study identified the effect of later school start times on academic performance in 1999–2006 data from middle school students in a large U.S. school district. Using variation in middle school start times both within and across schools, this analysis revealed that a delay in start times by 1 h lead to a 3-percentile point gain in both math and reading test scores for the average student. In the study's longitudinal analysis, the impact of delayed middle school start times on test scores persisted into the 10th grade with a larger effect for the lower end of the test score distribution.

Keeping in mind that being present in school is normally required for academic success, studies have examined sleep duration and attendance along with the effects of delaying school start times on attendance and tardiness. In a population-based study of over 8000 high school age adolescents, after adjusting for gender and socioeconomic status, short sleep duration and poor sleep efficiency were the sleep variables with the highest odds of non-attendance (Hysing, Haugland, Stormark, Boe, & Sivertsen, 2015). Likewise, improved absenteeism and particularly tardiness records stand out as one of the key findings in many of the school start time studies (e.g. Wolfson, Spaulding, Dandrow, & Baroni, 2007). For example, one study examined sleep patterns and school performance of early adolescents attending two urban, public middle schools with early (7:15 am) versus late (8:37 am) start times early and later in the year. At both times, students at the late-starting school had transcript-based tardiness records that were four times lower and reported waking up over 1 h later on school mornings, obtaining 50 min more sleep each night, and less daytime sleepiness than students at the early school (Wolfson et al., 2007).

In summary, the three reviews of non-experimental studies demonstrate that delaying school start times improves sleep and consequential daytime behaviors for middle and high school age adolescents. Furthermore, the studies in *Sleep Health's* special issue

(e.g. Troxel & Wolfson, 2017) on school start times and sleep explored some of the ongoing research questions including economic and legal questions and benefits, cross cultural questions, assessment, and implementation.

Undoubtedly, later school start times create the opportunity for adolescents to obtain sufficient sleep and to avoid social jet lag (i.e., experience like jet lag where one's regular sleep-wake schedule is misaligned with one's biological clock) with positive implications for academic performance. In turn, there is growing body of work that has looked directly at the impact of circadian timing and sleep duration on academic and cognitive performance. Studies have utilized both naturalistic, survey methodologies as well as experimental, laboratory-based approaches in a variety of research settings and from adolescents living in the U.S., as well as a wide range of other countries. Reviews of this scholarship continue to enhance our understanding of the findings, the limitations, and future research questions (Abraham & Scaria, 2015; Curcio, Ferrara, & De Gennaro, 2006; Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010; Tonetti, Natale, & Randler, 2015; Wolfson & Carskadon, 2003). Taking both approaches into account, findings demonstrate that poor sleep quality, daytime sleepiness, insufficient sleep, and evening circadian preference are correlated with decreased academic performance; sleep loss is associated with poor declarative and procedural learning; and laboratory-based sleep restriction might negatively affect neurobehavioral and cognitive performance (e.g., psychomotor reaction times, reduced awareness of the extent of sleep loss, impulsivity, and sustained attention (Curcio et al., 2006; Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001; Randazzo, Muehlbach, Schweitzer, & Walsh, 1998; Sadeh, Gruber, & Raviv, 2003).

In particular, studies have examined the associations between circadian preference, phase, cognitive functioning, and academic performance, including two meta-analytic reviews. Preckel and colleagues (Preckel, Lipnevich, Schneider, & Roberts, 2011) conducted four meta-analyses: morningness and cognitive ability measures (e.g., memory, processing speed verbal/quantitative reasoning), eveningness and cognitive ability, morningness and academic achievement (e.g., GPA), and eveningness and academic achievement. In the four analyses, associations between chronotype and cognitive ability as well as academic achievement were significant (Preckel et al., 2011). Eveningness was positively correlated with adolescents' cognitive ability, yet negatively associated with academic achievement measures. In contrast, morningness was negatively associated with cognitive ability and positively correlated with academic measures. Although a weak association, the authors suggest that evening types might be more cognitively adept and make use of a range of times to carry out their studies, particularly the late evening hours, a time when there are fewer distractions (Preckel et al., 2011). These findings, however, raise questions and require further research. In another meta-analysis, researchers examined circadian phase preference and academic performance (transcript, teacher or self-report GPA and/or standardized test scores) in studies that focused on “school” versus “university” age participants (Tonetti et al., 2015). Overall, there was a greater effect size in school-level compared to university-level studies. Also, self-report measures of grades revealed a stronger effect size in comparison to more objective measures. Similar to Preckel and colleagues' findings, this meta-analysis concluded that evening preference is associated with poorer academic performance, both in school and university populations. As the authors highlight, university students or older adolescents often have more independence and flexibility in creating their class schedule than middle or high school students allowing them to select classes that fit their own sleep-wake schedules, to avoid social jetlag, and to obtain more sleep (Tonetti et al., 2015). One clear limitation, however, is that it is unclear as to the age of the participants included in the studies categorized as “school” level in this systematic review.

There have been a few new and significant studies that engaged self-report methodologies, data base analyses, and experimental designs. Historically, sleep schedule regularity and academic performance has not been as well examined. Wolfson and Carskadon found that students who described themselves as struggling or failing school (C's, D's/F's) reported greater differences between their weekend and school-night sleep schedules (i.e., 2.3-h vs. 1.8-h difference between weekend and school-night bedtimes) than A and B students (Wolfson & Carskadon, 1998). Using a new approach to sleep regularity, Phillips and colleagues assessed 61 undergraduates for 30 days using sleep diaries and quantified sleep regularity using the sleep regularity index (Phillips et al., 2017). The sleep regularity index calculates the probability of an individual being asleep or awake at any two time-points 24 h apart, scaled so that an individual who is asleep or awake at the same times each day scores 100 versus an individual who sleeps randomly scores zero. Not surprising, a higher regularity index was associated with better academic performance (Phillips et al., 2017).

Using another novel approach, Smarr and Schirmer (Smarr & Schirmer, 2018) analyzed two years of data (N = 14,894) from a large US university's learning management system to examine the impact of misalignment between circadian rhythms and older adolescents' course schedules (i.e., social jet lag) on academic performance (GPA). After categorizing the students into “night owls,” “daytime finches,” and “morning larks,” based on their learning management system activity on non-class days, researchers compared their class times to their academic outcomes. Forty percent of the students learning management activity was synchronized with their class times; fifty percent were taking classes before they were fully alert, and 10 percent had already peaked by the time their classes started. Moreover, 60 percent of the students experienced more than 30 min of social jet lag, with greater social jetlag associated with lower semester GPAs, especially for owls (Smarr & Schirmer, 2018). In other words, when students' course schedules were aligned with their circadian schedule based on learning management activity, they were more likely to do better academically.

Recent experimental, laboratory-based studies further contribute to our understanding of the impact of sleep restriction, napping, and sleep timing on adolescents' cognitive functioning. Lo and colleagues simulated the school-night sleep loss and weekend night catch up common to many adolescents (Lo et al., 2017). In their design, they examined adolescents' neurobehavioral functioning during two periods of restricted and recovery sleep, as well as the effectiveness of afternoon naps in ameliorating neurobehavioral deficits associated with multiple nights of sleep restriction. Fifty-seven male adolescents (ages 15–19) experienced two cycles of 5 h time in bed for three nights with a 9-h time in bed recovery for two nights per cycle; half of the participants were given the opportunity for a 1-h afternoon nap following each sleep-restriction night. They found that participants in the non-nap condition showed a progressive decline in sustained attention, working memory, and speed of processing that did not return to baseline after two nights of recovery sleep. Napping attenuated but did not eliminate the adolescents' declining performance. In contrast, in Lo and

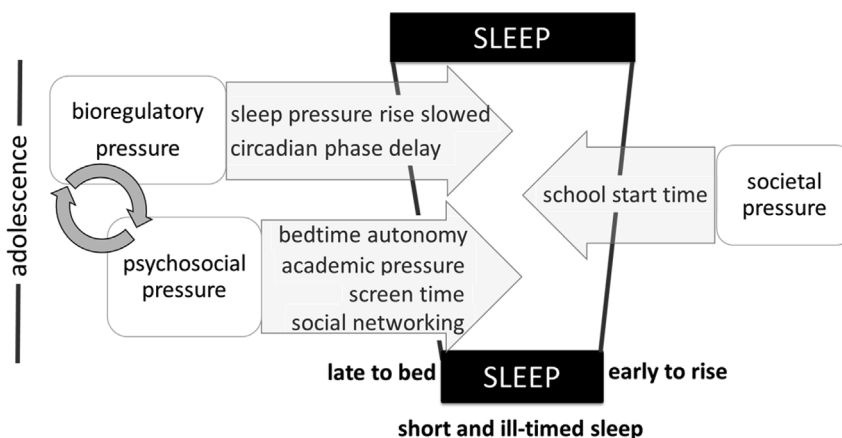
colleagues' previous study, they compared a similar group of adolescent males, randomly assigned to sleep restriction or control groups. Participants underwent a 2-week protocol consisting of three baseline nights (time in bed = 9 h), seven nights of sleep opportunity manipulation (time in bed = 5 h for sleep restriction and 9 h for controls) with three nights of recovery sleep (time in bed = 9 h) at a boarding school (Lo, Ong, Leong, Gooley, & Chee, 2016). In this case, the sleep restriction group demonstrated poorer sustained attention, working memory, and executive function; whereas, the control group was able to maintain cognitive performance levels.

Another recent study focused on a laboratory model for understanding the impact of cramming for exams instead of sleeping (Huang et al., 2016). In this case, researchers assessed the interaction of study timing and time in bed on vocabulary learning. Fifty-six adolescents (ages 15–19) were randomly assigned to a week of either 5-h (sleep restricted) or 9-h time in bed as part of a 14-day protocol at a boarding school. Participants studied 40 Graduate Record Examination (GRE)-type words on digital flashcards with word pairs spaced over four consecutive days or all at once during a single study session. Recall was examined immediately, 24 h, and 120 h (i.e., 5 days) after items were studied. For all retention intervals, recall was more impaired for adolescents in the sleep restriction condition. Cued recall on spaced items, however, was similar for the five and nine-hour time in bed groups. Paced learning seemed to mitigate the effects of sleep restriction on vocabulary recall, whereas students with insufficient sleep were more likely to forget items studied over short time intervals. Taken together, these experimental, laboratory studies demonstrate that for sleep-restricted adolescents, weekend “catch-up sleep,” even when combined with napping on school days, is inferior to obtaining sufficient and regular school-night sleep (Lo et al., 2016; Lo et al., 2017). Furthermore, adolescents with insufficient sleep who cram for exams might be particularly prone to having difficulty remembering new material; however, learning deficits might be minimized when students study over several days (Huang et al., 2016).

Finally, few studies have tried to improve adolescents' sleep hygiene practices and patterns as way of also improving or maintaining academic performance during middle school, a time when personal responsibility for learning and sleep habits is significant. Using a social learning model, the Sleep Smart Program aimed to improve sleep health behaviors and secondarily improve academic performance and behavioral well-being. A diverse group of seventh graders from two urban, middle schools were randomly assigned to a Sleep Smart program or a comparison group. In addition to improved sleep hygiene, more time in bed, and earlier bedtimes, sleep-smart participants also reported sustained academic performance over 7th grade whereas the comparison group's academic performance declined (Wolfson, Harkins, Johnson, & Marco, 2015).

### 3. The Perfect Storm: insufficient and ill-timed sleep and the troubling consequences

Fig. 1 illustrates a summary of the several factors discussed in this review that conspire to produce a *Perfect Storm* of insufficient and inappropriately-timed sleep. Bioregulatory pressures sustain evening alertness later into the night in maturing adolescents, at the same time parent-set bedtimes wane, academic demands increase, and social networks expand. Activities that are potentially stimulating or produce additional light stimuli can reinforce evening alertness and perpetuate a cycle of late sleep onsets as adolescents mature. Although there has been progress in recent years in the call for action to delay school start times, the major societal constraint on adolescents' sleep continues to be school start times. Other morning activities unique to individual families or cultures, such as religious activities or early sports practices, may also be considered. Although this review focused primarily on the school-related



**Fig. 1.** The Perfect Storm model, first presented by Carskadon (2011), illustrates several factors described in the text that contribute to changes in sleep behavior over the course of adolescent development. Bedtime and thus sleep onset is shifted later due to bioregulatory changes to the homeostatic sleep system and the circadian timing system. Psychosocial factors also contribute to delayed bedtimes. What we add here is the likely interaction between bioregulatory and psychosocial pressures (arrows). Changes to sleep physiology propping alertness later into the evening likely facilitates engaging in other activities besides sleep. Activities that are stimulating (e.g., engaging in their social network) or provide light exposure (e.g., from screens) during a time that delays circadian rhythms can exacerbate late sleep onset, but also feedback on the systems regulating sleep and wake. The length of sleep is not affected by these factors, it is only when societal pressures – the most notable being school start time – forces adolescents awake earlier than spontaneous arousal with an alarm clock.



negative consequences of this *Perfect Storm* of insufficient and ill-timed sleep, other serious consequences for adolescents include mood disturbances, behavioral problems, weight gain, and motor vehicle accidents.

Adolescence is an important developmental stage where sleep is strained both in duration and timing by a host of factors, some modifiable others not. Evidence for a relatively stable need for about 9 h of sleep per day on average, maturational modifications in bioregulatory processes, sleep homeostasis and circadian timing system, frame the nonmodifiable challenges. That said, appropriate lighting conditions (for example reduced light in the evening and increased light in the morning) can affect circadian timing in a positive way; however, feasible strategies to implement these circadian-based treatment strategies are still needed in this age group. Modifications of the school bell schedule can also loosen societal constraints on sleep duration and sleep irregularity, and many continue to work toward this goal. Moving forward, there is also a need to investigate these bioregulatory and psychosocial factors at an individual level to understand and identify individual differences in developmental trajectories that may predispose young people to more or less “stormy” sleep conditions. More work is also needed in identifying other exposures that could inform the *Perfect Storm* model, such as substance use (caffeine, alcohol, and marijuana), trauma, and economic disadvantage. Furthermore, molecular genetics including genotyping and epigenetic approaches may provide further insights into the bioregulatory processes. Thus, while the overall issues and concerns remain, the more that is learned through research supported by the National Institutes of Health or Center for Disease Control and Prevention in the US and other agencies and entities across the world, the better able adolescents, parents, families, teachers, administrators, pediatricians, and legislators will be to improve the sleep circumstances and sleep patterns of adolescents.

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